83605

STIC-Biotech/ChemLib

From: Chan, Christina
Sent: Sunday, January 05, 2003 11:47 AM
To: Canella, Karen; STIC-Biotech/ChemLib
Subject: RE: rush search request for 09/855,158

Please rush. Thanks Chris

Chris Chan
TC 1600 New Hire Training Coordinator and SPE 1644
308-3973
CM-1, 9B19

-----Original Message-----
From: **Canella, Karen**
Sent: Friday, January 03, 2003 9:37 PM
To: Chan, Christina
Subject: rush search request for 09/855,158

Chris,
Could you please authorize the following rush search for 09/855,158? It's an amended case due this bi-week.
Thanks,
Karen

Search and Interference Search
In the Protein Databases, the following peptides:
1. SEQ ID NO:6, 7, 13, 15 and 16

Karen Canella
office 9E17
mail 8E12
308-8362

POINT OF CONTACT:
PAUL SCHULWITZ
TECHNICAL INFO. SPECIALIST
CM1 6B06 TEL. (703) 305-1954

Searcher:_____
Phone:_____
Location:_____
Date Picked Up: 1/6
Date Completed: 1/8
Searcher Prep/Review:_____
Clerical:_____
Online time:_____

TYPE OF SEARCH:
NA Sequences:_____
AA Sequences:_____
Structures:_____
Bibliographic:_____
Litigation:_____
Full text:_____
Patent Family:_____
Other:_____

VENDOR/COST (where applic.)
STN:_____
DIALOG:_____
Questel/Orbit:_____
DRLink:_____
Lexis/Nexis:_____
Sequence Sys.:_____
WWW/Internet:_____
Other (specify):_____

L31 ANSWER 1 OF 1 MEDLINE on STN
ACCESSION NUMBER: 97477906 MEDLINE
DOCUMENT NUMBER: 97477906 PubMed ID: 9336839
TITLE: Ligand binding to proteins: the binding landscape model.
AUTHOR: **Miller D W**; Dill K A
CORPORATE SOURCE: Graduate Group in Biophysics, University of California at San Francisco 94143-1204, USA.
SOURCE: PROTEIN SCIENCE, **(1997 Oct) 6** (10) **2166-79**.
Journal code: 9211750. ISSN: 0961-8368.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980306
Last Updated on STN: 19980306
Entered Medline: 19980220

AB Models of ligand binding are often based on four assumptions: (1) steric fit: that binding is determined mainly by shape complementarity; (2) native binding: that ligands mainly bind to native states; (3) locality: that ligands perturb protein structures mainly at the binding site; and (4) continuity: that small changes in ligand or protein structure lead to small changes in binding affinity. Using a generalization of the 2D HP lattice model, we study ligand binding and explore these assumptions. We first validate the model by showing that it reproduces typical binding behaviors. We observe ligand-induced denaturation, ANS and heme-like binding, and "lock-and-key" and "induced-fit" specific binding behaviors characterized by Michaelis-Menten or more cooperative types of binding isotherms. We then explore cases where the model predicts violations of the standard assumptions. For example, very different binding modes can result from two ligands of identical shape. Ligands can sometimes bind highly denatured states more tightly than native states and yet have Michaelis-Menten isotherms. Even low-population binding to denatured states can cause changes in global stability, hydrogen-exchange rates, and thermal B-factors, contrary to expectations, but in agreement with experiments. We conclude that ligand binding, similar to protein folding, may be better described in terms of energy landscapes than in terms of simpler mass-action models.

L18 ANSWER 2 OF 3 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998066445 MEDLINE
DOCUMENT NUMBER: 98066445 PubMed ID: 9398215
TITLE: Determination of tumor necrosis factor binding protein disulfide structure: deviation of the fourth domain structure from the TNFR/NGFR family cysteine-rich region signature.
AUTHOR: Jones M D; Hunt J; Liu J L; Patterson S D; Kohno T; Lu H S
CORPORATE SOURCE: Department of Protein Structure, Amgen Inc., Amgen Center, Thousand Oaks, California 91320, USA.
SOURCE: BIOCHEMISTRY, **(1997 Dec 2)** 36 (48) 14914-23.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980122
Last Updated on STN: 20000303
Entered Medline: 19980108

AB Tumor necrosis factor binding protein is a soluble molecule derived from the extracellular domain of the 55 kDa human tumor necrosis factor receptor, which can block the biological function of tumor necrosis factor by **binding** to the growth factor. This **cysteine-rich** molecule is subdivided into four domains, each containing six conserved cysteines that form three intrachain disulfide linkages known as the **tumor necrosis factor** receptor/nerve growth factor receptor **family** cysteine-rich region signature structure. In an effort to elucidate the molecular integrity of the molecule, we performed detailed analysis and searched for strategies to elucidate the complete disulfide structure of the E. coli-derived tumor necrosis factor binding protein and to determine the disulfide arrangement in the fourth domain of Chinese hamster ovary cell-derived molecule. The methods employed included various proteolytic digestions, peptide mapping, partial reduction, and assignment of disulfides by N-terminal sequencing and matrix-assisted laser desorption ionization mass spectrometry with post-source decay. The first three domains of the molecule were confirmed to have disulfide structures identical to the cysteine-rich region signature structure found in the above-mentioned receptor superfamily. The fourth domain has a different structure from the first three domains where the last four cysteines form two disulfide bonds in opposite positions.

L1 ANSWER 1 OF 1 MEDLINE
ACCESSION NUMBER: 2000259066 MEDLINE
DOCUMENT NUMBER: 20259066 PubMed ID: 10801128
TITLE: TACI and BCMA are receptors for a TNF homologue implicated in B-cell autoimmune disease.
COMMENT: Comment in: Nature. 2000 Apr 27;404(6781):949-50
AUTHOR: Gross J A; Johnston J; Mudri S; Enselman R; Dillon S R; Madden K; Xu W; Parrish-Novak J; Foster D; Lofton-Day C; Moore M; Littau A; Grossman A; Haugen H; Foley K; Blumberg H; Harrison K; Kindsvogel W; Clegg C H
CORPORATE SOURCE: Department of Immunology, ZymoGenetics, Seattle, Washington 98102, USA.. grossj@zgi.com
SOURCE: NATURE, **(2000 Apr 27) 404** (6781) **995-9.**
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000606
Last Updated on STN: 20010716
Entered Medline: 20000525

AB B cells are important in the development of autoimmune disorders by mechanisms involving dysregulated polyclonal B-cell activation, production of pathogenic antibodies, and co-stimulation of autoreactive T cells. zTNF4 (BLyS, BAFF, TALL-1, THANK) is a member of the tumour necrosis factor (TNF) ligand family that is a potent co-activator of B cells in vitro and in vivo. Here we identify two receptors for zTNF4 and demonstrate a relationship between zTNF4 and autoimmune disease. Transgenic animals overexpressing zTNF4 in lymphoid cells develop symptoms characteristic of systemic lupus erythaematosus (SLE) and expand a rare population of splenic B-Ia lymphocytes. In addition, circulating zTNF4 is more abundant in NZBWF1 and MRL-lpr/lpr mice during the onset and progression of SLE. We have identified two TNF receptor family members, TACI and BCMA, that bind zTNF4. Treatment of NZBWF1 mice with soluble TACI-Ig fusion protein inhibits the development of proteinuria and prolongs survival of the animals. These findings demonstrate the involvement of zTNF4 and its receptors in the development of SLE and identify TACI-Ig as a promising treatment of autoimmune disease in humans.